Appendix H: Air Toxics

Introduction

Air toxics are defined as air pollutants other than those six criteria pollutants for which EPA sets acceptable concentrations in ambient air. The SARA 313 Toxic Release Inventory (TRI), covering 328 of the approximately 3000 potentially hazardous compounds detected in air, estimated that approximately 1.2 million tons of air toxics were released to the atmosphere in 1987 from U.S. stationary sources alone. While the TRI estimate tends to understate emissions of toxics for a number of reasons, it does show that large quantities of toxics are emitted into the atmosphere annually.

Effects of air toxics emissions are divided into three categories for study and assessment: cancer; "noncancer" effects, e.g. a wide variety of serious health effects such as abnormal development, birth defects, neurological impairment, or reproductive impairment, etc.; and ecological effects. Each year, these air toxics emissions contribute to significant adverse effects on human health, human welfare, and ecosystems. In EPA's 1987 *Unfinished Business Report*[1] cancer and noncancer air toxics risk estimates were considered sufficiently high, relative to risks addressed by other EPA programs, that the air toxics program area was among the few rated "high risk".

Limited Scope of this Assessment

The effects of air toxics emissions are difficult to quantify. The adverse health effects of toxics are often irreversible, not mitigated or eliminated by reduction in ongoing exposure, and involve particularly painful and/or protracted disease. Therefore these effects are not readily studied and quantified in human clinical studies, in contrast to, for example, ambient ozone. In addition, epidemiological studies of effects in exposed populations are often confounded by simultaneous exposure of subjects to a variety of pollutants. Therefore, the effects of air toxics are often quantified by extrapolating data from animal studies to human exposure and expressed as risk per unit of exposure. Incidence of noncancer effects, for example, often are difficult to translate into monetized benefits.

Similarly, the quantification of ecological effects due to emissions of air toxics is hampered by lack of sufficient information regarding contribution of sources to exposure, associations between exposure to mixtures of toxics and various ecological endpoints, and economic valuation for ecological endpoints.

The air toxics portion of this study is, of necessity, separate and more qualitative in nature than the benefit analysis conducted for the criteria air pollutants. Limitations in the quantitative analyses of air toxics effects led the Project Team to decide to exclude the available quantitative results from the primary analysis of CAA costs and benefits. Table H-1 presents the range of potential human health and ecological effects that can occur due to air toxics exposure. As indicated, this appendix presents quantitative estimates of benefits of CAA air toxics control for the cancer mortality endpoint for only nonutility stationary source and mobile source categories. Noncancer effects and ecological effects are described qualitatively.

[1] U.S. EPA. Office of Policy Planning and Evaluation. *Unfinished Business: A Comparative Assessment of Environmental Problems*. February 1987.

Table H-1. Health and Welfare Effects of Hazardous Air Pollutants.

Effect Category	Quantified Effects	Unquantified Effects	Other Possible Effects
Human Health	Cancer Mortality - nonutility stationary source - mobile source	Cancer Mortality - utility source - area source Noncancer effects - neurological - respiratory - reproductive - hematopoetic - developmental - immunological - organ toxicity	
Human Welfare		Decreased income and recreation opportunities due to fish advisories Odors	Decreased income resulting from decreased physical performance
Ecological		Effects on wildlife Effects on plants Ecosystem effects Loss of biological diversity	Effects on global climate
Other Welfare		Visibility Materials Damage	

History of Air Toxics Standards under the Clean Air Act of 1970

The 1970 Clean Air Act required the EPA to list a chemical as a hazardous air pollutant if it met the legislative definition provided:

> *"The term 'hazardous air pollutant' means an air pollutant to which no ambient air quality standard is applicable and which in the judgment of the Administrator may cause, or contribute to, an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness."*[2]

Once a HAP was listed, the EPA Administrator was required to:

> *"establish any such standard at the level which in his judgment provides an ample margin of safety to protect the public health from such hazardous air pollutant."*[3]

In other words the EPA had to first determine that a chemical was a HAP, and then regulate the emissions of each HAP based solely on human health effects and with an ample margin of safety. This regulatory mandate proved extremely difficult for EPA to fulfill, for reasons discussed below, and the result was that only seven HAPs were regulated over a period of 20 years.

Listing chemicals became a difficult task because of debates within and outside of the EPA surrounding issues of how much data are needed and which meth-

[2] 42 U.S.C. §1857(a)(1).

[3] 42 U.S.C. §1857(b).

odologies should be used to list a chemical as a HAP. An even more difficult issue was how to define the Congressional mandate to provide an "ample margin of safety." For carcinogens, there is generally no threshold of exposure considered to be without risk. What level of risk, then, is acceptable, and how should it be calculated? The EPA struggled to provide answers to these questions, and was challenged in court. The end result was a 1987 ruling by the D.C. Circuit Court that provided the EPA with a legal framework with which to determine an "ample margin of safety." This framework was interpreted and used by the EPA in its 1989 benzene regulations.

Quantifiable Stationary Source Air Toxics Benefits

One might be tempted to presume that the few federal HAP standards set would have achieved relatively substantial reductions in quantifiable risk. While some standards set under section 112 of the Clean Air Act appear to have achieved significant reductions in cancer incidence, the coverage, quantification, and monetization of the full range of potential adverse effects remains severely limited. This fact serves to highlight the inadequacy of current methods of evaluating HAP control benefits. This limited ability to estimate the total human health and ecological benefits of HAP reductions is an important area for future research. Thus the quantifiable benefits for CAA air toxics control presented here are limited in scope.

There are three sources of information that provide a picture of potential stationary source air toxics benefits of the CAA. EPA's Cancer Risk studies attempted to broadly assess the magnitude and nature of the air toxics problem by developing quantitative estimates of cancer risks posed by selected air toxics and their sources. Secondly, risk assessments conducted in conjunction with the promulgation of National Emissions Standards for Hazardous Air Pollutants (NESHAPs) offer a snapshot of potential monetized cancer mortality benefits. Finally, the Project Team attempted to estimate historical non-utility stationary source HAP-related direct inhalation cancer incidence reductions. Results from each of these studies are presented below.

EPA Analyses of Cancer Risks from Selected Air Toxic Pollutants

The Agency conducted two efforts to broadly assess the magnitude and nature of the air toxics problem. The 1985 report entitled, "The Air Toxics Problem in the United States: An Analysis of Cancer Risks for Selected Pollutants"[4] otherwise known as the "Six Month Study," was intended to serve as a "scoping" study to provide a quick assessment of the air toxics problem utilizing only readily available data on compound potencies, emissions, and ambient pollutant concentrations. The Agency updated this analysis of cancer risks in the 1990 report entitled "Cancer Risk from Outdoor Exposure to Air Toxics" referred to here as the "1990 Cancer Risk study."[5]

For the pollutant and source categories examined, the 1990 Cancer Risk study estimated the total nationwide cancer incidence due to outdoor concentrations of air toxics to range from 1,700 to as many as 2,700 excess cancer cases per year, with 14 compounds accounting for approximately 95 percent of the annual cancer cases. Additionally, point sources contribute 25 percent of annual cases and area sources contribute 75 percent of annual cases. Mobile sources account for 56 percent of the nationwide total.[6]

The Six Month study indicates that the criteria air pollutant programs appear to have done more to reduce air toxics levels during the 1970 to 1990 period than have regulatory actions aimed at specific toxic compounds promulgated during the same period. Metals and polynuclear compounds usually are emitted as particulate matter and most of the volatile organic compounds are ozone precursors. As such, they are regulated under State Implementation Plan (SIP) and New Source Performance Standard (NSPS) programs and Title II motor vehicle regulations. A number of reports cited indicate significant reductions in air toxics emissions attributable to actions taken un-

[4] U.S. EPA. Office of Air Quality Planning and Standards. *The Air Toxics Problem in the United States: An Analysis of Cancer Risks for Selected Pollutants*. May 1985. EPA-450/1-85-001.

[5] U.S. EPA. Office of Air Quality Planning and Standards. *Cancer Risk from Outdoor Exposure to Air Toxics*. September 1990. EPA-450/1-90-004a.

[6] The 1990 Cancer Risk study reported approximately 500 - 900 more cancer cases per year than the Six Month Study due primarily to the inclusion of more pollutants, better accounting of emissions sources, and, in some cases, increases in unit risk estimates.

der SIP, NSPS and mobile source programs. Additionally, EPA conducted a comparison of air quality and emissions data for 1970 with the estimates of cancer incidence for 1980.[7] Methods, assumptions and pollutants included were held constant over the period. The analysis showed a significant decrease in incidence during the decade due to improvements in air quality, presumably related to general regulatory programs. For the 16 pollutants studied, estimated nationwide cancer incidence decreased from 3600 in 1970 to 1600 in 1980. The 1990 Cancer Risk Study did not attempt to update this analysis.

Although it is difficult to draw quantitative conclusions from these two studies regarding the benefits of CAA air toxics control, it is apparent that the pollutant-specific and source category-specific NESHAPs were not structured to reduce significant air toxic emissions from area and mobile sources. In fact, the 1990 Cancer Risk Study indicates that considerable cancer risk remained prior to passage of the 1990 CAA Amendments: as many as 2,700 excess cancer cases annually. Some studies indicate that the criteria air pollutant program played a critical role during the 1970 to 1990 period in achieving air toxic emission reductions and therefore decreasing cancer risk.

Cancer Risk Estimates from NESHAP Risk Assessments

In looking back at the estimated effects of the HAP standards, EPA found that the effects of the NESHAPs were not quantified completely. These estimates occurred at a time when emission estimation and risk assessment methodologies for HAPs were first being developed. One consequence is that because emissions were not fully characterized, air toxics exposures could not be completely assessed. Additionally, most assessments only focused on the specific HAP being listed under the CAA and did not assess the reduction of other pollutants, which are currently considered HAPs. For example, while the vinyl chloride standard reduces emissions of ethylene dichloride, these emission reductions were not assessed in the risk assessment. In a different context, reductions of HAP may also achieve reductions of VOC and PM. The benefits of such reductions generally were also not evaluated. In addition, EPA generally did not assess the potential exposure to high, short-term concentrations of HAP and therefore did not know whether toxic effects from acute exposures would have been predicted and possibly addressed by the HAP standards.

In addition, people living near emission sources of concern are often exposed to a mix of pollutants at once. Some pollutants have been shown to act synergistically together to create a health risk greater than the risk that would be expected by simply adding the two exposure levels together. More research is needed to understand the effects of multiple-pollutant exposures. Finally, HAP risks tend to be distributed unevenly across exposed populations, with particularly high exposures occurring closest to emission sources. It should be noted that HAP exposure to specific populations may tend to fall disproportionately among the poor and minorities, who are more likely to live in close proximity to emitting facilities.

With the above caveats in mind, Table H-2 provides information about maximum individual risk taken from the Federal Register notices for the NESHAPs promulgated before the 1990 amendments to the Clean Air Act. The benefits are calculated by multiplying the estimated annual incidence reduction by the $4.8 million valuation per statistical life (1990 dollars). These benefit estimates provide a snapshot of potential monetized benefits for the year in which each NESHAP was promulgated. Of course these estimates do not include air toxics benefits for other health and ecological benefit categories, or air toxics benefits from co-control of criteria air pollutants. All uncertainties associated with the original estimates remain.

Non-utility Stationary Source Cancer Incidence Reductions

The Project Team commissioned two studies to estimate reductions in cancer incidence due to pre-1990 NESHAPs: the PES Study and the ICF Re-analysis. The methodology used for most air pollutant evaluations involved a “back calculation” for the estimation of incidence reductions. However, the EPA has elected not to rely on the results of this analysis given critical methodological flaws. Despite the Project Team’s concerns, the methodology and results of the two studies are presented below in the interest of full disclosure and to guide efforts to develop a more valid

[7] Hunt, W.F., Faoro, R.B. and Curran, T.C., “Estimation of Cancer Incidence Cases and Rates for Selected Toxic Air Pollutants Using Ambient Air Pollution Data, 1970 vs. 1980”. U.S. EPA. April 1985.

Table H-2. Cancer Incidence Reductions and Monetized Benefits for NESHAPs.

Pollutant	Source Category	Year Promulgated	Pre-Reg Maximum Individual Risk	Post-Reg Maximum Individual Risk	Reduction in Cancer Incidence (per year)	Benefits in $million per year (1990$)
benzene		1985	$1.5x10^{-3}$	$4.5x10^{-4}$	.31	1.5
benzene	coke by-product	1984	$7x10^{-3}$	$2x10^{-4}$	1.95	9.4
benzene	storage vessels	1982	$4.5x10^{-4}$	$3x10^{-5}$	0.01 to 0.06	0.05 to 0.3
benzene	waste operations	1986	$2x10^{-3}$	$5x10^{-5}$	0.55	2.6
benzene	transfer operations	1987	$6x10^{-3}$	$4x10^{-5}$	0.98	4.7
arsenic	primary copper	1986	$1.3x10^{-3}$ to $5x10^{-6}$	$1.2x10^{-3}$ to $3x10^{-6}$	0.09	0.4
arsenic	glass manuf.	1986	$7x10^{-4}$ to $3x10^{-5}$	$1.7x10^{-4}$ to $6x10^{-6}$	0.117 to 0.0034	0.02 to 0.6
asbestos	demolition	1973			100	480
vinyl chloride	PVC production	1975			10.5	50.4

and reliable analysis of the health-related benefits of HAP reductions in the upcoming section 812 Prospective studies.

PES Study

Methodology

The first attempt to estimate, for this study, historical non-utility stationary source HAP-related direct inhalation cancer incidence reductions was conducted by Pacific Environmental Services (PES). The basic approach used in the PES study was to adjust the cancer incidence estimates developed for EPA's 1990 Cancer Risk study to reflect the changes in emissions of, and exposures to, 14 key HAPs: arsenic, asbestos, benzene, 1,3-butadiene, carbon tetrachloride, chloroform, hexavalent chromium, dioxin, ethylene dichloride, ethylene dibromide, formaldehyde, gasoline vapors, products of incomplete combustion (PICs), and vinyl chloride.

The first step was to compile baseline incidence levels, defined as cancer cases per million population, for each of the 14 pollutants. The point estimates of incidence from the 1990 Cancer Risk study were used for this purpose. For some source categories, the "best point estimate" from the 1990 Cancer Risk study was used, for others a mid-point was selected.[8] These baseline incidence levels were based on measured ambient concentrations of the pollutant, modeled concentrations, or both.

The second step involved allocating baseline incidence levels to the individual source categories known to emit the relevant pollutant. In some cases,

[8] For some of the source categories, the original NESHAP/Air Toxic Exposure and Risk Information System (NESHAP/ATERIS) estimates of incidence were not available, in which case the baseline incidence was obtained from the 1989 National Air Toxics Information Clearinghouse(NATICH) Database Report. (See PES, "Draft Summary of Methodology Used for Cancer from Stationary Sources," memorandum from Ken Meardon, PES to Vasu Kilaru, US EPA, March 22, 1993, p. 2.)

adjustments were made to reflect differences among the vintages of source category-specific data.[9] All baseline incidence estimates were ultimately expressed relative to a 1985 base year.[10] The assumption was then made that source-category incidence rates were proportional to the level of emissions from that source category.

Next, levels of control for each source category-specific incidence rate were estimated for each of the target years of the present analysis (i.e., 1970, 1975, 1980, 1985, and 1990).[11] Source category-specific activity level indicators were then established and linked to changes in corresponding activity indicators provided by the J/W macroeconomic modeling results. Activity levels were estimated for each source category, for each of the target years, and for each of the two scenarios.

Finally, source category/pollutant combination incidence levels for both the control and no-control scenarios were developed. These incidence levels were developed based on the baseline incidence levels, the activity indicators, and the control levels for each year. Both of these latter two factors varied between the control and no-control scenarios. The activity levels differed based on the specific levels of related sector economic activity predicted by the J/W model for the control and no-control scenario. The control levels prevailing in each of the target years were used for the control scenario, and the 1970 control level was applied throughout the 1970 to 1990 period for the no-control scenario.[12] The formula used for these calculations was as follows:[13]

$$I_{ty} = I_{by} \times [\frac{A_{ty}}{A_{by}}] \times [\frac{P_{ty}}{P_{by}}] \times [\frac{(1 - C_{ty})}{(1 - C_{by})}] \qquad (1)$$

where:

- I = cancer incidence for a source category-pollutant combination
- A = activity level for a source category
- P = population
- C = control level for a source category-pollutant combination
- ty = target year (1970 ... 1990)
- by = base year

Findings

The PES analysis concluded that substantial reductions in HAP-related cancer cases were achieved during the reference period of the present study. The vast majority of these estimated reductions were attributable to reduced exposures to asbestos, particularly from manufacturing and fabricating sources.[14] In fact, roughly 75 percent of the total reduction in cancer cases averaged over the 1970 to 1990 period were attributed to asbestos control.[15] Figure H-1 summarizes the PES study overall cancer incidence reductions and the relative contribution of asbestos-related reductions over the study period.

The Project Team had several concerns about the PES results. First and foremost, the reductions in asbestos-related cancer cases appeared to be substantially higher than expected, particularly in the earlier target years. Second, the control scenario activity level indicators for several sources with which Project Team members were familiar did not appear to be even remotely consistent with actual historical activity patterns.[16] Finally, the level of documentation of the analytical methodologies, assumptions, and results was insufficient to ascertain the validity and reliability of

[9] For example, six discrete sources for vinyl chloride were identified in the Six-Month Study Update. Point estimate incidences for each of these source categories came from separate references with databases corresponding to different years. (See PES, "retrospective analysis for section 812(a) Benefits Study," September 30, 1992, p. 8.)

[10] See PES, March 22, 1993 memorandum, p. 3.

[11] Control level estimates were based on one of the following: control efficiencies for related criteria pollutants defined in the criteria pollutant analysis, reference documents such as Control Technology Guidelines (CTGs) or Background Information Documents (BIDs), preambles for related regulations, or EPA experts. (See PES, March 22, 1993 memorandum, p. 3.)

[12] More detailed descriptions of the methodology and associated uncertainties are provided in "Retrospective Analysis for section 812(a) Benefits Study," a September 30, 1992 memorandum from Ken Meardon, PES to Vasu Kilaru, US EPA.

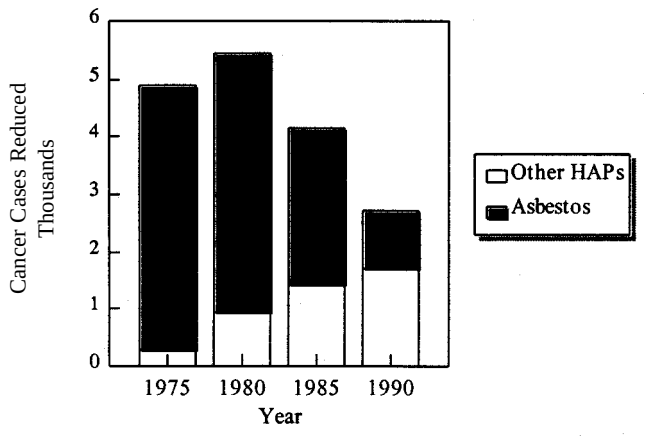
[13] See PES, March 22, 1993 memorandum, p. 4.

[14] PES, "Cancer Risk Estimates from Stationary Sources," memorandum from Ken Meardon, PES to Vasu Kilaru, US EPA, March 5, 1993.

[15] ICF, "Direct Inhalation Incidence Benefits," Draft Report, November 11, 1994, p. 10.

[16] For example, the activity indicators for Municipal Waste Combustors (MWCs) incorporated in the PES analysis decline dramatically throughout the 1975 to 1990 period. (See PES, March 5, 1993 memorandum to Vasu Kilaru, p. 10). In reality, overall MWC capacity and throughput increased significantly over this period.

Figure H-1. PES Estimated Reductions in HAP-Related Cancer Cases.



the results. Ultimately, the Project Team determined that it was necessary to conduct a formal review and re-analysis of the cancer incidence reductions associated with non-utility stationary source HAP controls. The results of the PES analysis remain a relevant part of the record of the present study, however, since they provided a substantial basis for the subsequent re-analysis by ICF Incorporated.

ICF Re-analysis

Methodology

The purposes of the ICF Re-analysis were to examine the methodology and results of the PES study, particularly to address the aforementioned concerns of the Project Team, and to develop a revised set of estimates. Due to significant constraints on the resources remaining for HAP analysis in the section 812 study, however, only a few key HAPs could be investigated in depth and many important issues could not be addressed.[17] Furthermore, the effects of two early and potentially important HAP standards –the Beryllium and Mercury NESHAPs– could not be evaluated. Nevertheless, the ICF Re-analysis clarified some potential sources of uncertainty in the PES results and provided revised cancer incidence reduction estimates for several HAPs.

A key uncertainty in the PES results was associated with the use of a "back-calculation" technique to estimate incidence reductions for some HAPs. The back-calculation technique estimates uncontrolled incidence by dividing residual incidence by the assumed control efficiency. This approach means uncontrolled incidence, and therefore incidence reductions, are highly sensitive to small changes in assumed control efficiency.[18] In some cases, the PES analysis may have used control efficiencies which were too high, resulting in overestimation of uncontrolled incidence and therefore incidence reductions attributable to the CAA.[19] The vinyl chloride incidence reduction estimates appear to be significantly influenced by the use of this back-calculation technique. Another important source of uncertainty identified by ICF involved the potential overestimation of incidence totals when source apportionment is based on measured ambient concentrations.[20] ICF was unable, however, to perform an extensive evaluation of the activity level indicators used in the PES study.[21]

The first step undertaken in the re-analysis was to conduct a screening test to identify the HAPs which accounted for the most significant estimated incidence reductions. Based on this screening analysis, ICF eliminated 1,3-butadiene, carbon tetrachloride, chloroform, gasoline vapors, chromium, formaldehyde, and PICs from the detailed re-analysis effort.

Detailed reviews were then conducted for the remaining HAPs: vinyl chloride, dioxins, ethylene dibromide (EDB), ethylene dichloride (EDC), benzene, asbestos, and arsenic. In the re-analysis of these HAPs, ICF determined whether a forward- or back-calculation technique was used for the relevant source categories of a given HAP, reviewed the regulatory

[17] For example, the Project Team sought to develop and apply a methodology for estimating a central tendency estimate for the total carcinogenic risk imposed by all the HAPs examined. The intent was to address concerns about potential overestimation of aggregate risk measures when combining upper bound risk estimates of multiple HAPs. Unfortunately, resources were insufficient to continue development of this methodology.

[18] An example of this back-calculation technique illustrating the sensitivity to the assumed control efficiency is presented on page 12 of the draft ICF report.

[19] See ICF Draft Report, p. 12.

[20] See ICF Draft Report, p. 9.

[21] See ICF Draft Report, p. 13.

history of the relevant source categories to re-evaluate the assumed control efficiencies, and reviewed the upper-bound unit risk factor for each HAP. Revised total incidence reduction estimates for each HAP and for each target year were then calculated using the same basic calculation procedure used by PES. Finally, ICF identified a number of residual deficiencies in the analysis which could only be addressed through additional research and analysis.[22]

Findings

The ICF Re-analysis largely affirmed the original results obtained by PES; primarily because the PES analysis itself served as the basis for the re-analysis and only minor adjustments were adopted for many critical variables. In particular, most Project Team concerns regarding the PES methodology could not be resolved, including uncertainties associated with activity levels, assumed control efficiencies, and the unexpectedly high estimated incidence reductions associated with asbestos. In fact, the ICF Re-analysis produced a revised upper bound estimate for vinyl chloride-related incidence reductions which were even higher than the asbestos benefits.

Several sets of results were developed by ICF and presented in either the November 1994 draft report or in briefing materials prepared for the Science Advisory Board Clean Air Act Compliance Analysis Council Physical Effects Subcommittee (SAB/ACCACAPERS) in May 1995. The first set of results is based on the assumption of 100 percent source compliance with HAP control requirements. An alternative set of results was developed assuming an 80 percent compliance rate with applicable standards. Given the linear effect of changes in compliance rates, these results were precisely 20 percent lower than the first set of estimates. At the May 1995 ACCACAPERS briefing, estimates based on the 100 percent compliance estimates were presented. For asbestos, the revised incidence reductions were presented and characterized as upper bound. The asbestos estimates were then combined with upper and lower bound estimates for vinyl chloride and for "all other compounds." Figure H-2 presents the total cancer incidence reductions derived from the ICF Re-analysis, using the asbestos estimates combined with the lower bound estimates for non-asbestos HAPs.

Figure H-2. ICF Estimated Reductions in Total HAP-Related Cancer Cases Using Upper Bound Asbestos Incidence and Lower Bound Non-Asbestos HAP Incidence.

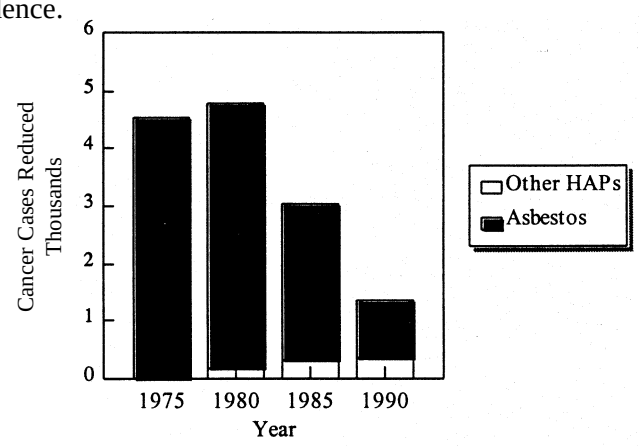


Figure H-3. ICF Estimated Reduction in Total HAP-Related Cancer Cases Using Upper Bound Incidence for All HAPs.

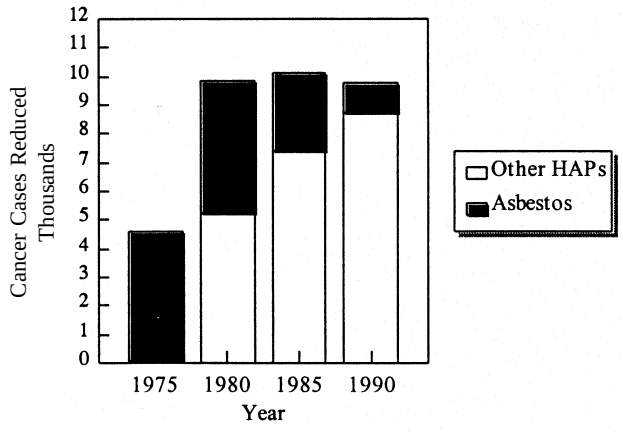


Figure H-3 presents a comparable compilation reflecting the upper bound estimates for all HAPs.

The Project Team remains concerned about these incidence reduction estimates, particularly given the doubts raised by the SAB/ACCACAPERS at the May 1995 presentation of these results. For instance, several critical assumptions are needed to make this analysis valid when applied to EPA's NESHAPs. The flaws in these assumptions are described below.

(1) The risk estimates described in the 1990 Cancer Risk study, which served as the baseline for determining risk reductions, were accepted without question. There are myriad uncertainties in these estimates

[22] Additional details of the ICF Re-analysis methodology can be found in ICF, "Direct Inhalation Incidence Benefits," Draft Report, November 11, 1994.

that must be recognized, as the study was designed only to generate rough order-of-magnitude estimates of the extent of the air toxics cancer problem.

(2) The percent control efficiency for emission reductions, which is calculated in each standard, would have to apply across every facility. Typically, the emissions reductions vary between facilities; using a single average reduction could skew the results.

(3) There is a direct correlation between the number of tons of emissions reduced and incidence reduced by a specific regulation. Given the assumption of a linear, non-threshold dose-response curve (as is typically done for cancer), this is theoretically correct.

(4) Finally, the back calculation approach assumes that there is 100 percent compliance with the regulation.

EPA staff reviewed the "back calculation" approach for one of the more controversial aspects of the vinyl chloride (VC) NESHAP. The PES study estimates benefits at 426 cases reduced in 1990. The ICF Re-analysis resulted in an even higher estimate, between 1,000 and 7,000 cases annually. An analysis by EPA staff indicated that these vinyl chloride risk estimates are highly suspect given historical cancer incidence data for hepatic angiocarcinoma, a specific cancer that has been linked to vinyl chloride (Koppikar and Fegley, 1995). The following analysis demonstrates the inadequacies of the assumptions in the 1993 study.

(1) In the actual standard, no control technology was required for emissions from oxychlorination vents at ethylene dichloride (EDC)/VC plants. Applying "back calculation" for these emissions is inappropriate.

(2) In 1985, there were an estimated 8,000 fabrication plants which processed resins produced by PVC plants, thus resulting in VC emissions, which were exempt from the VC NESHAP. They emit very small quantities of VC and back calculation is not appropriate.

(3) The 1993 study uses a baseline estimate of 18 residual cases from the NESHAP/ATERIS data base. There is no evidence that these cases resulted only from emissions from PVC and EDC/VC plants.

(4) The risk analysis performed for the October 21, 1976 final VC regulation projected an incidence reduction of 11 cases per year.

In contrast, the PES study, using the "back calculation" method derived the following annual incidence reductions:

1980 - 250 cases
1985 - 360 cases
1990 - 430 cases

The subsequent back calculation conducted in the ICF Re-analysis resulted in incidence reductions as much as an order of magnitude higher than these.

Even considering the slightly different industrial output assumptions imposed by macroeconomic modeling, such a stark contrast is difficult to explain except for a critically flawed approach. Growth in activity and population nor other factors explain the difference in these two estimates. Given that the same general methodology was used for all of the air toxic pollutant assessments as was used for the VC NESHAP evaluation, there is reason to believe that cancer incidence results for the other air toxic pollutants are also flawed.

Mobile Source HAP Exposure Reductions

EPA's Cancer Risk report estimated that approximately 60 percent of the total carcinogenic risk posed by HAPs was attributed to mobile sources, with stationary sources contributing 15 percent and area sources contributing the remaining 25 percent.[23] The relative importance of mobile sources to total HAP exposure was a significant motivation behind EPA's subsequent effort to examine exposures and risks from mobile source HAPs.[24] Although available analytical resources were severely limited, the Project Team nevertheless decided it was necessary to perform at least an initial screening analysis to estimate the differences in mobile source HAP exposures between the control and no-control scenarios configured for the present study.

[23] Cancer Risk report, Page ES-12.

[24] See US EPA/OAR/OMS, "Motor Vehicle-Related Air Toxics Study," EPA 420-R-93-005. April 1993.

Methodology

The approach used by ICF/SAI in conducting the mobile source HAP analysis closely followed the approach used in the EPA Motor Vehicle-Related Air Toxics Study (MVATS).[25] Recognizing the dearth of HAP ambient concentration and exposure data, both studies use carbon monoxide (CO) concentrations as the basis for estimating mobile source HAP concentrations and exposures. An important difference between the two studies, however, is that the ICF/SAI study adjusted the estimated change in ambient CO concentrations to take account of background[26] and non-mobile source[27] CO emissions. The HAP exposure function used in the ICF/SAI analysis is summarized by the following equation:

$$E = ((C \times A) - B) \times S \times M \times \frac{(VOC \times HAP)}{CO} \quad (2)$$

where :

- E = exposure to motor vehicle-emitted HAP
- C = annual ambient CO concentration to annual CO exposure concentration conversion factor
- A = county-level annual average ambient CO concentration
- B = background CO concentration
- S = no-control to control scenario CO concentration adjustment factor (equals 1 for the control scenario)
- M = total CO exposure to mobile source CO exposure conversion factor
- VOC = VOC emissions by year, county, and scenario
- HAP = VOC speciation factor by mobile source HAP
- CO = CO emissions by year, county, and scenario

Details of the derivation of each of the variables applied in the above equation are provided in the ICF/SAI report. However, in essence, the calculation involves the following basic steps.

First, annual average county-level CO ambient monitoring data are compiled from the EPA Aerometric Information Retrieval System (AIRS) database. After adjusting for background and non-mobile source contributions, these annual average ambient CO concentrations are converted to annual average CO exposure concentrations. As in the EPA MVATS, this conversion is made based on the Hazardous Air Pollutant Exposure Model - Mobile Sources (HAPEM-MS) population exposure model, which takes account of time spent in five indoor and outdoor microenvironments: indoors at home, other indoor, in-vehicle, outdoors near roadway, and other outdoor.[28] After adjusting for CO exposures attributable to non-mobile sources of CO, the CO exposures are converted to exposures for each of the mobile source HAPs based on available VOC speciation data and the ratio of co-located VOC and CO emissions.[29] These calculations are repeated for the no-control scenario after adjusting for differences in CO ambient concentrations for each target year and for differences in fuel composition.

Results

By 1990, CAA controls resulted in significant reductions in exposure to motor vehicle HAPs. Figure H-4 summarizes the nationwide annual average exposure levels, in micrograms per cubic meter, for each of the five HAPs analyzed under the control and no-control scenarios. Additional detailed results, including breakdown by urban versus rural environments and comparisons with the EPA MVATS estimates, are provided in the ICF/SAI report.

Analytical resources to carry forward these exposure estimates to derive estimates of the changes in motor vehicle HAP-related adverse effects attributable to historical CAA programs were not available.

[25] ICF/SAI, "Retrospective Analysis of Inhalation Exposure to Hazardous Air Pollutants from Motor Vehicles," October 1995, p. 4.

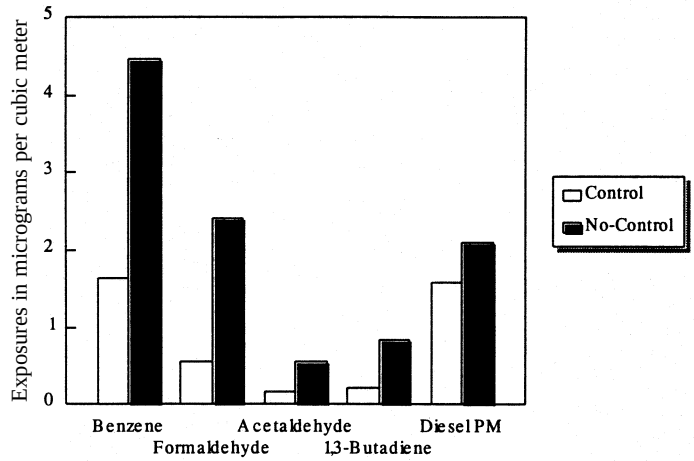
[26] Background CO is produced by the oxidation of biogenic hydrocarbons. See ICF/SAI, p. 7.

[27] The EPA MVATS attributed all measured CO to motor vehicles, resulting in an overestimation of motor-vehicle contributions to CO concentration changes. See ICF/SAI, p. 8. The MVATS assumption would also lead to a subsequent overestimation of changes in HAP exposures.

[28] See ICF/SAI, p. 3.

[29] The same HAP emission fractions used in the EPA MVATS were used herein, except for diesel PM which is not proportional to VOC emissions. Instead, diesel PM emission factors were developed using year-specific PART5 diesel PM emission factors and VMT estimates for diesel-powered vehicles.

Figure H-4. National Annual Average Motor Vehicle HAP Exposures (µg/m3).



Non-Cancer Health Effects

Broad gaps exist in the current state of knowledge about the quantifiable effects of air toxics exposure. This is particularly true for a wide range of health effects such as tumors, abnormal development, birth defects, neurological impairment, or reproductive impairment, etc. For example, the EPA's Non-Cancer Study[30] found that ambient concentrations for a substantial number of monitored and modeled HAPs exceeded one or more health benchmarks.[31] However no accepted methodology exists to quantify the effects of such exceedences. More data on health effects is needed for a broad range of chemicals.

Ecological Effects

Through the 1970s and 1980s, the adverse effects of toxic pollution on the Great Lakes became clear and undeniable. Over the same time period, scientists began collecting a convincing body of evidence that toxic chemicals released to the air can travel long distances and be deposited on land or water far from the original sources. An example of this evidence is the presence of such contaminants as PCBs, toxaphene, and other pesticides in fish in Lake Siskiwit, a lake on an island on upper Lake Superior, which has no waterborne sources of pollution. Toxaphene, a pesticide used primarily in the southeastern U.S. cotton belt, has been found as far away as the Arctic, with a decreasing air concentration gradient from the southeast toward the Great Lakes and the north Atlantic regions.

Similarly, a growing body of evidence showed that pollutants that were persistent (do not easily break down) and bioaccumulating (not significantly eliminated from the body) were magnifying up the food chain, such that top predator fish contained levels up to millions of times greater than the harmless levels in the water. As such, those who ate those large fish, such as humans, eagles, mink, and beluga whales could receive very high exposures to the pollutants. Wildlife were beginning to show adverse effects in the wild, that could be duplicated in the lab. In the Great Lakes, such chemicals as PCBs, mercury, dieldrin, hexachlorobenzene, Lindane, lead compounds, cadmium compounds, DDT/DDE, and others are of significant concern. In other places in the country, similar effects are being experienced, especially with mercury, which is transported primarily by air, but exposure to which is primarily through contaminated fish. It was this kind of information about DDT and toxaphene that led to their being banned in the U.S. under FIFRA.

While ecological and economical sciences are not yet sufficiently advanced to support the kind of comprehensive, quantitative evaluation of benefits needed for the present study, selected local and regional scale adverse ecological effects of HAPs, and their adverse consequences for human health and welfare, can and have been surveyed. In May 1994, the EPA issued its first "Report to Congress on Deposition of Air Pollutants to the Great Waters."[32] The Great Waters Report examined the pollutants contributing to adverse ecological effects, the potential significance of the contribution to pollutant loadings from deposition of airborne pollutants, and the potential adverse effects associated with these pollutant loadings. Key HAPs identified in the Great Waters Report include PCBs, mercury, dioxins, and other heavy metals and toxic organics.

[30] U.S. Environmental Protection Agency, "Toxic Air Pollutants and Noncancer Risks: Screening Studies," External Review Draft, September, 1990.

[31] Relevant benchmarks include Acceptable Daily Intake (ADI), the estimate of daily exposure at which adverse health effects are unlikely; and Lowest Observed Actual Effect Level (LOAEL), which is the lowest exposure level at which significant adverse health effects are observed.

[32] USEPA/OAR/OAQPS, "Deposition of Air Pollutants to the Great Waters, First Report to Congress," EPA-453/R-93-055, May 1994.

Of particular relevance to the present assessment, the Great Waters Report demonstrated the significance of transport and transformation of HAPs through food webs, leading to increased toxicity and biomagnification. A prime example of adverse transport and transformation is mercury. Transformation from inorganic to methylated forms significantly increases the toxic effects of mercury in ecosystems. A prime example of biomagnification is PCBs. As noted in the Great Waters Report:

> *"Pollutants of concern [such as PCBs] accumulate in body tissues and magnify up the food web, with each level accumulating the toxics from its diet and passing the burden along to the animal in the next level of the food web. Top consumers in the food web, usually consumers of large fish, may accumulate chemical concentrations many millions of times greater than the concentrations present in the water...High risk groups...include breast-feeding mothers because breast-fed babies continue to accumulate [pollutants] from their mothers after birth. For example, they can have PCB levels four times higher than their mothers after six to nine months of breast feeding."*[33]

Because of the risk of significant exposure to infants and other high-risk groups, such as "sport anglers, Native Americans, and the urban poor,"[34] a substantial number of fish consumption advisories have been issued in recent years. Current fish advisories for the Great Lakes alone include widespread advisories for PCB's, chlordane, mercury and others, cautioning that nursing mothers, pregnant women, women who anticipate bearing children, female children of any age and male children age 15 and under not eat certain high-food chain fish species. It should be noted as well that 40 states have issued mercury advisories in some freshwater bodies, and nine states have issued mercury advisories for every freshwater waterbody in the state (these states are Maine, New Hampshire, Vermont, Massachusetts, New York, New Jersey, Missouri, Michigan, and Florida).

There is little evidence indicating that the CAA had much beneficial effect on air toxic deposition to water bodies. Since the early NESHAPs were based on direct inhalation, primarily cancer effects close to a plant, they did not address the issue of cumulative effects of persistent pollutants far from the source. It was for this reason that section 112(m) was included in the 1990 CAA Amendments, with requirements to study and document the atmospheric contribution of water pollutants, the adverse human health and environmental effects resulting and the sources that should be controlled to prevent adverse effects, and additionally, to promulgate regulations to prevent adverse effects.

Conclusions — Research Needs

As has been demonstrated, there are broad gaps in the current state of knowledge about the quantifiable effects of air toxics exposure for a wide range of both human health and environmental effects. The following discussion outlines areas in which further research is needed in order to adequately quantify the benefits of air toxics control.

Health Effects

- Develop health effects data on pollutants for which limited or no data currently exists. Such studies should be focused on pollutants with a relatively high probability of exposure and/or potential adverse health effects.

- Understand mechanism of action of pollutants, for example through pharmacokinetic modeling. This will allow for a more accurate assessment of the effects of these pollutants on humans.

- Conduct research on factors that affect variations in susceptibility of human populations and determine the distribution of these factors in the U.S.

- Conduct research to better understand interactive effects of multiple pollutant exposures.

- Develop methodologies to derive alternative estimates of human cancer risk from existing upper-bound methods.

- Acquire data and develop dose-response relationships for critical noncancer effects such as developmental, neurotoxic, mutagenic, res-

[33] EPA-453/R-93-055, May 1994, p. ix.

[34] EPA-453/R-93-055, May 1994, p. x.

piratory and other effects. In particular, design methodology to quantify effects of exposures above health benchmarks.

- Acquire data and develop methods to estimate effects from acute exposure.

Exposure Assessment

- Expand data collection efforts: pre- and post-control emissions; HAP speciation; facilities location; facility parameters (stack heights, distances from stacks to fencelines, etc.).

- Develop more comprehensive exposure models which incorporate activity patterns, indirect exposures, total body burden, ratios of time spent indoors to outdoors.

- Continue to refine uncertainty analysis methods.

Ecosystem Effects

- Reliable estimates/measures of the levels of persistent bioaccumulating toxics in different media (air, water column, soils and sediments)

- Work to correlate levels of persistent bioaccumulating toxics with exposures, biota concentrations/accumulation, and adverse effects, especially subtle effects such as wasting, behavioral effects, and developmental effects.

- Criteria for effects, such as a wildlife correlate to a RfD or dose-response curve. This work should be done to complement the mass balance efforts now being completed, which will model source emissions to water column concentrations, then design research to predict effects on living resources given those predicted levels.

- Work to determine the effects of mixtures of persistent bioaccumulating toxic pollutants, and to determine cause-effect relationships of exposures over long periods of time.

- Studies to evaluate toxic effects in less well understood terrestrial systems such as: soil organisms/invertebrates, food web effects, amphibian effects, effects on endangered species and phytotoxic effects.

- Work to improve understanding of effects of toxic air pollutants on wetland species and wetland functions.

Economic Valuation

- Develop valuation estimates for endpoints for which inadequate estimates currently exist. These valuation estimates must be consistent with the kinds of damages expected.

- Initiate broad-scope economic valuation of air toxics program using survey techniques.

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